

WEST Search History

DATE: Thursday, December 07, 2006

| <u>Hide?</u> | <u>Set Name</u> | <u>Query</u> | <u>Hit Count</u> |
|---|-----------------|--------------|------------------|
| <i>DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI; PLUR=YES; OP=ADJ</i> | | | |

| | | | |
|--------------------------|-----|------------------------|-----|
| <input type="checkbox"/> | L11 | L10 and @py<1998 | 0 |
| <input type="checkbox"/> | L10 | L8 and pharmacological | 117 |
| <input type="checkbox"/> | L9 | L8 and pharmacologica | 0 |
| <input type="checkbox"/> | L8 | L7 and alzheimer | 190 |
| <input type="checkbox"/> | L7 | L6 and cross linking | 225 |
| <input type="checkbox"/> | L6 | redox and amyloid | 786 |

| | | | |
|---|--|--|--|
| <i>DB=DWPI,JPAB,EPAB,USOC,USPT,PGPB; PLUR=YES; OP=ADJ</i> | | | |
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| | | | |
|--------------------------|----|------------------|----|
| <input type="checkbox"/> | L5 | TANZI-RUDOLPH-E! | 49 |
| <input type="checkbox"/> | L4 | TANZI-RUDOLPH-E! | 49 |
| <input type="checkbox"/> | L3 | ATWOOD-CRAIG-S! | 7 |
| <input type="checkbox"/> | L2 | HUANG-XUDONG! | 18 |
| <input type="checkbox"/> | L1 | BUSH-ASHLEY-I! | 23 |

END OF SEARCH HISTORY

Can # 10/643, 226
WEST (PGPB, USPT, USOC, EPAB, JPAB, DWPI)
12/07/06 AD

FILE 'MEDLINE' ENTERED AT 16:25:29 ON 07 DEC 2006

FILE 'BIOSIS' ENTERED AT 16:25:29 ON 07 DEC 2006

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=> s redox and cross linking
L1 380 REDOX AND CROSS LINKING

=> s l1 and Alzheimer
L2 6 L1 AND ALZHEIMER

=> s l1 and pharmacologic
L3 4 L1 AND PHARMACOLOGIC

=> s l2 and l3
L4 0 L2 AND L3

=> disp l2 ibib abs 1-6

L2 ANSWER 1 OF 6 MEDLINE on STN

ACCESSION NUMBER: 2004433062 MEDLINE

DOCUMENT NUMBER: PubMed ID: 15231727

TITLE: Tyrosine gated electron transfer is key to the toxic mechanism of Alzheimer's disease beta-amyloid.

AUTHOR: Barnham Kevin J; Haeffner Fredrik; Ciccotosto Giuseppe D; Curtain Cyril C; Tew Deborah; Mavros Christine; Beyreuther Konrad; Carrington Darryl; Masters Colin L; Cherny Robert A; Cappai Roberto; Bush Ashley I

CORPORATE SOURCE: Department of Pathology, University of Melbourne, Victoria, Australia.. kbarnham@unimelb.edu.au

CONTRACT NUMBER: R01AG12686 (NIA)

SOURCE: The FASEB journal : official publication of the Federation of American Societies for Experimental Biology, (2004 Sep) Vol. 18, No. 12, pp. 1427-9. Electronic Publication: 2004-07-01.

Journal code: 8804484. E-ISSN: 1530-6860.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200504

ENTRY DATE: Entered STN: 2 Sep 2004

Last Updated on STN: 6 Apr 2005

Entered Medline: 5 Apr 2005

AB Alzheimer's disease (AD) is characterized by the presence of neurofibrillary tangles and amyloid plaques, which are abnormal protein deposits. The major constituent of the plaques is the neurotoxic beta-amyloid peptide (Abeta); the genetics of familial AD support a direct role for this peptide in AD. Abeta neurotoxicity is linked to hydrogen peroxide formation. Abeta coordinates the redox active transition metals, copper and iron, to catalytically generate reactive oxygen species. The chemical mechanism underlying this process is not well defined. With the use of density functional theory calculations to delineate the chemical mechanisms that drive the catalytic production of H2O2 by Abeta/Cu, tyrosine10 (Y10) was identified as a pivotal residue for this reaction to proceed. The relative stability of tyrosyl radicals facilitates the electron transfers that are required to drive the reaction. Confirming the theoretical results, mutation of the tyrosine residue to alanine inhibited H2O2 production, Cu-induced radicalization, dityrosine cross-linking, and neurotoxicity.

L2 ANSWER 2 OF 6 MEDLINE on STN

ACCESSION NUMBER: 2000395121 MEDLINE

DOCUMENT NUMBER: PubMed ID: 10866821

Can#10/64328
STN(MEDLINE, 10/10)
AD
12/7/06

TITLE: Transition metal-mediated glycoxidation accelerates cross-linking of beta-amyloid peptide.
AUTHOR: Loske C; Gerdemann A; Schepl W; Wycislo M; Schinzel R; Palm D; Riederer P; Munch G
CORPORATE SOURCE: Physiological Chemistry I, Biocenter, University of Wurzburg, Germany.
SOURCE: European journal of biochemistry / FEBS, (2000 Jul) Vol. 267, No. 13, pp. 4171-8.
PUB. COUNTRY: GERMANY: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 24 Aug 2000
Last Updated on STN: 24 Aug 2000
Entered Medline: 16 Aug 2000

AB beta-Amyloid deposits, hallmarks of Alzheimer's disease, contain both sugar-derived 'advanced glycation end products' (AGEs) and copper and iron ions. Our *in vitro* experiments using synthetic beta-amyloid peptide and glucose or fructose show that formation of covalently cross-linked high-molecular-mass beta-amyloid peptide oligomers is accelerated by micromolar amounts of copper (Cu^+ , Cu^{2+}) and iron (Fe^{2+} , Fe^{3+}) ions. Formation of these covalent AGE cross-links can be inhibited by capping agents of amino groups, redox-inactive metal chelators and antioxidants, suggesting that these drugs may be able to slow down the formation of insoluble beta-amyloid deposits *in vivo* and possibly the progression of Alzheimer's disease.

L2 ANSWER 3 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:413611 BIOSIS
DOCUMENT NUMBER: PREV200400411706
TITLE: Tyrosine gated electron transfer is key to the toxic mechanism of Alzheimer's disease beta-amyloid.
AUTHOR(S): Barnham, Kevin J. [Reprint Author]; Haeffner, Fredrik; Ciccotosto, Giuseppe D.; Curtain, Cyril C.; Tew, Deborah; Mavros, Christine; Beyreuther, Konrad; Carrington, Darryl; Masters, Colin L.; Cherny, Robert A.; Cappai, Roberto; Bush, Ashley I.
CORPORATE SOURCE: Dept Pathol, Univ Melbourne, Parkville, Vic, 3010, Australia
kbarnham@unimelb.edu.au; bush@helix.mgh.harvard.edu
SOURCE: FASEB Journal, (July 2004) Vol. 18, No. 10. print.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 27 Oct 2004
Last Updated on STN: 27 Oct 2004

AB Alzheimer's disease (AD) is characterized by the presence of neurofibrillary tangles and amyloid plaques, which are abnormal protein deposits. The major constituent of the plaques is the neurotoxic beta-amyloid peptide (Abeta); the genetics of familial AD support a direct role for this peptide in AD. Abeta neurotoxicity is linked to hydrogen peroxide formation. It coordinates the redox active transition metals, copper and iron, to catalytically generate reactive oxygen species. The chemical mechanism underlying this process is not well defined. With the use of density functional theory calculations to delineate the chemical mechanisms that drive the catalytic production of H₂O₂ by Abeta/Cu, tyrosine10 (Y10) was identified as a pivotal residue for this reaction to proceed. The relative stability of tyrosyl radicals facilitates the electron transfers that are required to drive the reaction. Confirming the theoretical results, mutation of the tyrosine residue to alanine inhibited H₂O₂ production, Cu-induced radicalization, dityrosine cross-linking, and neurotoxicity.

L2 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2004:145826 BIOSIS
DOCUMENT NUMBER: PREV200400145645
TITLE: Modulators of LRP - mediated Abeta clearance.
AUTHOR(S): Moir, R. D. [Reprint Author]; Tseitlin, K. A. [Reprint Author]; Bush, A. I. [Reprint Author]; Huang, X. [Reprint Author]; Tanzi, R. E. [Reprint Author]
CORPORATE SOURCE: Dept. of Neurol., Genet. and Aging Unit, Massachusetts Gen. Hosp., Charlestown, MA, USA
SOURCE: Society for Neuroscience Abstract Viewer and Itinerary Planner, (2003) Vol. 2003, pp. Abstract No. 772.10.
http://sfn.scholarone.com. e-file.
Meeting Info.: 33rd Annual Meeting of the Society of Neuroscience. New Orleans, LA, USA. November 08-12, 2003.
Society of Neuroscience.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004

AB Accumulation of the Abeta protein as beta-amyloid deposits is the pathological hallmark of Alzheimer's disease (AD). A significant fraction of both the soluble and insoluble pools of Abeta in AD brain are cross-linked dimeric/trimeric species. Cross-linking is one of several chemical modifications that Abeta can undergo under the mild redox conditions of AD brain parenchyma. The physiochemical properties, toxicity and clearance pathways for these species is poorly characterized. However, evidence is accumulating that these redox modified species may play central roles in the pathological aggregation and neurotoxic properties of Abeta. The low density lipoprotein receptor-related protein (LRP) is a multiligand cell surface receptor that mediates Abeta clearance. Abeta does not bind the receptor directly but first forms a complex with the LRP ligands apolipoprotein E (APOE) or alpha-2-macroglobulin (a2M). The Abeta/chaperone complexes then bind to LRP and are internalized. We have previously presented data showing redox modified Abeta has reduced binding for APOE and a2M. We have extended these findings and more fully characterized the inhibited binding of APOE and a2M to SDS stable dimeric species of Abeta and the resulting attenuated LRP-mediated clearance of these forms. Our most recent findings have also identified copper and zinc as modulators of Abeta/chaperone binding. In addition, we will present data on the altered physiochemical properties of redox generated dimeric/trimeric Abeta species. Our data supports a role for redox generated dimers as "seeds" for the aggregation of monomeric normal Abeta. The 40 residue Abeta isomer (Abeta40) preferentially forms cross-linked dimers while SDS stable trimeric complexes are more readily generated by the 42 amino acid form of the peptide (Abeta42). SDS stable heterodimers of Abeta40 and Abeta42 readily form under mild redox conditions when both protein isoforms are present.

L2 ANSWER 5 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2001:244340 BIOSIS
DOCUMENT NUMBER: PREV200100244340
TITLE: The structure and enzyme-like activity of the free radical site generated in the glyoxal-mediated cross-linked proteins.
AUTHOR(S): Lee, Hong In [Reprint author]; Yim, Moon Bin [Reprint author]; Chock, P. Boon [Reprint author]; Stadtman, Earl R. [Reprint author]
CORPORATE SOURCE: NHLBI, NIH, 3 Center Drive, MSC-0342, Bethesda, MD, 20892-0342, USA
SOURCE: FASEB Journal, (March 7, 2001) Vol. 15, No. 4, pp. A180.

print.

Meeting Info.: Annual Meeting of the Federation of American Societies for Experimental Biology on Experimental Biology 2001. Orlando, Florida, USA. March 31-April 04, 2001.
CODEN: FAJOEC. ISSN: 0892-6638.

DOCUMENT TYPE:

Conference; (Meeting)

Conference; Abstract; (Meeting Abstract)

LANGUAGE:

English

ENTRY DATE:

Entered STN: 23 May 2001

Last Updated on STN: 19 Feb 2002

AB During the glycation reaction between reducing sugars and free amino groups of proteins, alpha-dicarbonyl compounds, such as glyoxal, methylglyoxal, and deoxyglucosones, are produced. These compounds are more reactive than the parent sugars for reacting-with amino groups of proteins to form inter-and intra-molecular cross-links of proteins and stable advanced end products that are known to accumulate with aging, diabetes mellitus, Alzheimer's disease, and other diseases. In this study, we investigated the structure and redox properties of cross-linked amino acids and proteins produced by glyoxal. Model reactions between glyoxal or glycolaldehyde and the amino acids, alanine or N-alpha-acetyl-lysine, produced free radicals. The structure of this radical was identified by EPR spectroscopy as N-substituted pyrazine radical cation, which was formed by cross-linking two amino acids. Glycation of BSA by these carbonyl compounds also generated stable protein-bound free radical species, probably the N-substituted pyrazine radical cation as observed with amino acids. The glycated protein reduced ferricytochrome c to ferrocytochrome c, which was accompanied by a large increase in the EPR signal amplitude of the protein-bound free radical cation. In addition, the glycated protein catalyzed the oxidation of ascorbate. These results indicate that protein glycation generates active centers for catalyzing one electron redox reactions. One of the active centers generated by glyoxal is the cross-linked N-substituted pyrazine and its radical cation.

L2 ANSWER 6 OF 6 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:359752 BIOSIS

DOCUMENT NUMBER: PREV200000359752

TITLE:

Transition metal-mediated glycoxidation accelerates cross-linking of beta-amyloid peptide.

AUTHOR(S):

Loske, Claudia; Gerdemann, Andrea; Schepl, Walter; Wycislo, Matthias; Schinzel, Reinhard; Palm, Dieter; Riederer, Peter; Muench, Gerald [Reprint author]

CORPORATE SOURCE:

IZKF Nachwuchsgruppe Neurowissenschaften, Liebigstrasse 27, 04103, Leipzig, Germany

SOURCE:

European Journal of Biochemistry, (July, 2000) Vol. 267, No. 13, pp. 4171-4178. print.

CODEN: EJBCAI. ISSN: 0014-2956.

DOCUMENT TYPE:

Article

LANGUAGE:

English

ENTRY DATE:

Entered STN: 23 Aug 2000

Last Updated on STN: 8 Jan 2002

AB beta-Amyloid deposits, hallmarks of Alzheimer's disease, contain both sugar-derived 'advanced glycation end products' (AGES) and copper and iron ions. Our in vitro experiments using synthetic beta-amyloid peptide and glucose or fructose show that formation of covalently cross-linked high-molecular-mass beta-amyloid peptide oligomers is accelerated by micromolar amounts of copper (Cu⁺, Cu²⁺) and iron (Fe²⁺, Fe³⁺) ions. Formation of these covalent AGE cross-links can be inhibited by capping agents of amino groups, redox-inactive metal chelators and antioxidants, suggesting that these drugs may be able to slow down the formation of insoluble beta-amyloid deposits in vivo and possibly the progression of Alzheimer's disease.

=> disp l3 ibib abs 1-4

L3 ANSWER 1 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2003366230 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12899942
TITLE: T cell receptor-stimulated generation of hydrogen peroxide
inhibits MEK-ERK activation and lck serine phosphorylation.
AUTHOR: Kwon J; Devadas S; Williams M S
CORPORATE SOURCE: Immunology Department, Jerome H. Holland Laboratory for the
Biomedical Sciences, American Red Cross, Rockville, MD,
USA.
SOURCE: Free radical biology & medicine, (2003 Aug 15) Vol. 35, No.
4, pp. 406-17.
Journal code: 8709159. ISSN: 0891-5849.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200408
ENTRY DATE: Entered STN: 6 Aug 2003
Last Updated on STN: 25 Jul 2004
Entered Medline: 10 Aug 2004

AB Previous studies indicated that antigen receptor (TcR) stimulation of mature T cells induced rapid generation of reactive oxygen species (ROS). The goal of the current study was to examine the role(s) of ROS in TcR signal transduction, with a focus upon the redox-sensitive MAPK family. TcR cross-linking of primary human T blasts and Jurkat human T cells rapidly activated the ERK, JNK, p38 and Akt kinases within minutes, and was temporally associated with TcR-stimulated production of hydrogen peroxide (H₂O₂). TcR-induced activation of ERK was selectively augmented and sustained in the presence of pharmacologic antioxidants that can quench or inhibit H₂O₂ production (NAC, MnTBAP and Ebselen, but not DPI), while activation of JNK and Akt were largely unaffected. This was paralleled by concurrent changes in MEK1/2 phosphorylation, suggesting that ROS acted upstream of MEK-ERK activation. Molecular targeting of H₂O₂ by overexpression of peroxiredoxin II, a thioredoxin dependent peroxidase, also increased and sustained ERK and MEK activation upon TcR cross-linking. Enhancement of ERK phosphorylation by antioxidants correlated with increased and sustained serine phosphorylation of the src-family kinase lck, a known ERK substrate. Thus, the data suggest that TcR-stimulated production of hydrogen peroxide negatively feeds back to dampen antigen-stimulated ERK activation and this redox-dependent regulation may serve to modulate key steps in TcR signaling.

L3 ANSWER 2 OF 4 MEDLINE on STN
ACCESSION NUMBER: 2002074881 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11781366
TITLE: Discrete generation of superoxide and hydrogen peroxide by T cell receptor stimulation: selective regulation of mitogen-activated protein kinase activation and fas ligand expression.
AUTHOR: Devadas Satish; Zaritskaya Luba; Rhee Sue Goo; Oberley
Larry; Williams Mark S
CORPORATE SOURCE: Department of Immunology, Holland Laboratory, American Red Cross, Rockville, MD 20855, USA.
SOURCE: The Journal of experimental medicine, (2002 Jan 7) Vol. 195, No. 1, pp. 59-70.
Journal code: 2985109R. ISSN: 0022-1007.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201

ENTRY DATE: Entered STN: 25 Jan 2002
Last Updated on STN: 1 Feb 2002
Entered Medline: 31 Jan 2002

AB Receptor-stimulated generation of reactive oxygen species (ROS) has been shown to regulate signal transduction, and previous studies have suggested that T cell receptor (TCR) signals may involve or be sensitive to ROS. In this study, we have shown for the first time that TCR cross-linking induced rapid (within 15 min) generation of both hydrogen peroxide and superoxide anion, as defined with oxidation-sensitive dyes, selective pharmacologic antioxidants, and overexpression of specific antioxidant enzymes. Furthermore, the data suggest the novel observation that superoxide anion and hydrogen peroxide are produced separately by distinct TCR-stimulated pathways. Unexpectedly, TCR-stimulated activation of the Fas ligand (FasL) promoter and subsequent cell death was dependent upon superoxide anion, but independent of hydrogen peroxide, while nuclear factor of activated T cells (NFAT) activation or interleukin 2 transcription was independent of all ROS. Anti-CD3 induced phosphorylation of extracellular signal-regulated kinase (ERK) 1/2 required hydrogen peroxide generation but was unaffected by superoxide anion. Thus, antigen receptor signaling induces generation of discrete species of oxidants that selectively regulate two distinct redox sensitive pathways, a proapoptotic (FasL) and a proliferative pathway (ERK).

L3 ANSWER 3 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:477876 BIOSIS
DOCUMENT NUMBER: PREV200300477876
TITLE: T cell receptor-stimulated generation of hydrogen peroxide inhibits MEK-ERK activation and lck serine phosphorylation.
AUTHOR(S): Kwon, J.; Devadas, S.; Williams, M. S. [Reprint Author]
CORPORATE SOURCE: Dept. of Immunology, Holland Laboratory, American Red Cross, 15601 Crabbs Branch Way, Rockville, MD, 20855, USA willmark@usa.redcross.org
SOURCE: Free Radical Biology & Medicine, (August 15 2003) Vol. 35, No. 4, pp. 406-417. print.
ISSN: 0891-5849 (ISSN print).

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Oct 2003
Last Updated on STN: 15 Oct 2003

AB Previous studies indicated that antigen receptor (TcR) stimulation of mature T cells induced rapid generation of reactive oxygen species (ROS). The goal of the current study was to examine the role(s) of ROS in TcR signal transduction, with a focus upon the redox-sensitive MAPK family. TcR cross-linking of primary human T blasts and Jurkat human T cells rapidly activated the ERK, JNK, p38 and Akt kinases within minutes, and was temporally associated with TcR-stimulated production of hydrogen peroxide (H2O2). TcR-induced activation of ERK was selectively augmented and sustained in the presence of pharmacologic antioxidants that can quench or inhibit H2O2 production (NAC, MnTBAP and Ebselen, but not DPI), while activation of JNK and Akt were largely unaffected. This was paralleled by concurrent changes in MEK1/2 phosphorylation, suggesting that ROS acted upstream of MEK-ERK activation. Molecular targeting of H2O2 by overexpression of peroxiredoxin II, a thioredoxin dependent peroxidase, also increased and sustained ERK and MEK activation upon TcR cross-linking. Enhancement of ERK phosphorylation by antioxidants correlated with increased and sustained serine phosphorylation of the src-family kinase lck, a known ERK substrate. Thus, the data suggest that TcR-stimulated production of hydrogen peroxide negatively feeds back to dampen antigen-stimulated ERK activation and this redox-dependent regulation may serve to modulate key steps in TcR signaling.

L3 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2002:130237 BIOSIS
DOCUMENT NUMBER: PREV200200130237
TITLE: Discrete generation of superoxide and hydrogen peroxide by T cell receptor stimulation: Selective regulation of mitogen-activated protein kinase activation and Fas ligand expression.
AUTHOR(S): Devadas, Satish; Zaritskaya, Luba; Rhee, Sue Goo; Oberley, Larry; Williams, Mark S. [Reprint author]
CORPORATE SOURCE: Department of Immunology, Holland Lab, American Red Cross, 15601 Crabbs Branch Way, Rockville, MD, 20855, USA
willmark@usa.redcross.org
SOURCE: Journal of Experimental Medicine, (January 7, 2002) Vol. 195, No. 1, pp. 59-70. print.
CODEN: JEMEAV. ISSN: 0022-1007.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Feb 2002
Last Updated on STN: 26 Feb 2002
AB Receptor-stimulated generation of reactive oxygen species (ROS) has been shown to regulate signal transduction, and previous studies have suggested that T cell receptor (TCR) signals may involve or be sensitive to ROS. In this study, we have shown for the first time that TCR cross-linking induced rapid (within 15 min) generation of both hydrogen peroxide and superoxide anion, as defined with oxidation-sensitive dyes, selective pharmacologic antioxidants, and overexpression of specific antioxidant enzymes. Furthermore, the data suggest the novel observation that superoxide anion and hydrogen peroxide are produced separately by distinct TCR-stimulated pathways. Unexpectedly, TCR-stimulated activation of the Fas ligand (FasL) promoter and subsequent cell death was dependent upon superoxide anion, but independent of hydrogen peroxide, while nuclear factor of activated T cells (NFAT) activation or interleukin 2 transcription was independent of all ROS. Anti-CD3 induced phosphorylation of extracellular signal-regulated kinase (ERK) 1/2 required hydrogen peroxide generation but was unaffected by superoxide anion. Thus, antigen receptor signaling induces generation of discrete species of oxidants that selectively regulate two distinct redox sensitive pathways, a proapoptotic (FasL) and a proliferative pathway (ERK).

=>

LE 'CAPLUS' ENTERED AT 16:31:35 ON 07 DEC 2006
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FILE COVERS 1907 - 7 Dec 2006 VOL 145 ISS 24
FILE LAST UPDATED: 6 Dec 2006 (20061206/ED)

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<http://www.cas.org/infopolicy.html>

=> E BUSH ASHLEY I/IN 25
E1 1 BUSH ARTHUR B JR/IN
E2 1 BUSH ASHLEY/IN
E3 10 --> BUSH ASHLEY I/IN
E4 3 BUSH ASHLEY IAN/IN
E5 1 BUSH ASHLEY L/IN
E6 1 BUSH BRADLEY S/IN
E7 1 BUSH BRADLEY STEPHEN/IN
E8 1 BUSH BRIAN/IN
E9 2 BUSH BRIAN DAVID/IN
E10 1 BUSH C ALLEN/IN
E11 1 BUSH CAROL L/IN
E12 1 BUSH CATHERINE/IN
E13 1 BUSH CHARLENE ELEANOR/IN
E14 8 BUSH CHARLES N/IN
E15 2 BUSH CHARLES NEAL/IN
E16 2 BUSH CHRISTOPHER DAVID/IN
E17 1 BUSH CHRISTOPHER JOHN THOMSON/IN
E18 1 BUSH CHRISTOPHER N/IN
E19 3 BUSH CONDON S/IN
E20 1 BUSH CRAIG P/IN
E21 2 BUSH CRAIG PALMER/IN
E22 1 BUSH DARRELL/IN
E23 1 BUSH DARRELL C/IN
E24 24 BUSH DAVID/IN
E25 1 BUSH DAVID A/IN

=> S (E3) AND (REDOX REACTIVE METAL, ALZHEIMER, AMYLOID BETA PEPTIDE, CROSSLINKING)
10 "BUSH ASHLEY I"/IN
133466 REDOX
8 REDOXES
133469 REDOX
(REDOX OR REDOXES)
300155 REACTIVE
151 REACTIVES
300260 REACTIVE
(REACTIVE OR REACTIVES)
1697487 METAL
855839 METALS
2059434 METAL

(METAL OR METALS)
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
25384 AMYLOID
1706 AMYLOIDS
25476 AMYLOID
(AMYLOID OR AMYLOIDS)
1418613 BETA
1327 BETAS
1418689 BETA
(BETA OR BETAS)
361103 PEPTIDE
264219 PEPTIDES
462369 PEPTIDE
(PEPTIDE OR PEPTIDES)
197031 CROSSLINKING
128 CROSSLINKINGS
197086 CROSSLINKING
(CROSSLINKING OR CROSSLINKINGS)
0 REDOX REACTIVE METAL, ALZHEIMER, AMYLOID BETA PEPTIDE, CROSSLINKING

(REDOX (W) REACTIVE (W) METAL (W) ALZHEIMER (W) AMYLOID (W) BETA (W) PEPTIDE (W) CROSSLINKING)
L1 0 ("BUSH ASHLEY I"/IN) AND (REDOX REACTIVE METAL, ALZHEIMER, AMYLOID
BETA PEPTIDE, CROSSLINKING)

=> S (E3) AND (REDOX REACTIVE METAL, ALZHEIMER)
10 "BUSH ASHLEY I"/IN
133466 REDOX
8 REDOXES
133469 REDOX
(REDOX OR REDOXES)
300155 REACTIVE
151 REACTIVES
300260 REACTIVE
(REACTIVE OR REACTIVES)
1697487 METAL
855839 METALS
2059434 METAL
(METAL OR METALS)
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
0 REDOX REACTIVE METAL, ALZHEIMER
(REDOX (W) REACTIVE (W) METAL (W) ALZHEIMER)
L2 0 ("BUSH ASHLEY I"/IN) AND (REDOX REACTIVE METAL, ALZHEIMER)

=> S (E3) AND (REDOX METAL, ALZHEIMER)
10 "BUSH ASHLEY I"/IN
133466 REDOX
8 REDOXES
133469 REDOX
(REDOX OR REDOXES)
1697487 METAL
855839 METALS
2059434 METAL
(METAL OR METALS)
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
0 REDOX METAL, ALZHEIMER

(REDOX (W) METAL (W) ALZHEIMER)
 L3 0 ("BUSH ASHLEY I"/IN) AND (REDOX METAL, ALZHEIMER)

 => S (E3) AND (REDOX , ALZHEIMER)
 10 "BUSH ASHLEY I"/IN
 133466 REDOX
 8 REDOXES
 133469 REDOX
 (REDOX OR REDOXES)
 42377 ALZHEIMER
 3013 ALZHEIMERS
 42445 ALZHEIMER
 (ALZHEIMER OR ALZHEIMERS)
 1 REDOX , ALZHEIMER
 (REDOX (W) ALZHEIMER)

 L4 0 ("BUSH ASHLEY I"/IN) AND (REDOX , ALZHEIMER)

 => S (E3) AND (ALZHEIMER)
 10 "BUSH ASHLEY I"/IN
 42377 ALZHEIMER
 3013 ALZHEIMERS
 42445 ALZHEIMER
 (ALZHEIMER OR ALZHEIMERS)

 L5 7 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER)

 => S (E3) AND (ALZHEIMER)
 10 "BUSH ASHLEY I"/IN
 42377 ALZHEIMER
 3013 ALZHEIMERS
 42445 ALZHEIMER
 (ALZHEIMER OR ALZHEIMERS)

 L6 7 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER)

 => E BUSH ASHLEY I/IN 25

 E1 1 BUSH ARTHUR B JR/IN
 E2 1 BUSH ASHLEY/IN
 E3 10 --> BUSH ASHLEY I/IN
 E4 3 BUSH ASHLEY IAN/IN
 E5 1 BUSH ASHLEY L/IN
 E6 1 BUSH BRADLEY S/IN
 E7 1 BUSH BRADLEY STEPHEN/IN
 E8 1 BUSH BRIAN/IN
 E9 2 BUSH BRIAN DAVID/IN
 E10 1 BUSH C ALLEN/IN
 E11 1 BUSH CAROL L/IN
 E12 1 BUSH CATHERINE/IN
 E13 1 BUSH CHARLENE ELEANOR/IN
 E14 8 BUSH CHARLES N/IN
 E15 2 BUSH CHARLES NEAL/IN
 E16 2 BUSH CHRISTOPHER DAVID/IN
 E17 1 BUSH CHRISTOPHER JOHN THOMSON/IN
 E18 1 BUSH CHRISTOPHER N/IN
 E19 3 BUSH CONDON S/IN
 E20 1 BUSH CRAIG P/IN
 E21 2 BUSH CRAIG PALMER/IN
 E22 1 BUSH DARRELL/IN
 E23 1 BUSH DARRELL C/IN
 E24 24 BUSH DAVID/IN
 E25 1 BUSH DAVID A/IN

 => S (E3) AND (ALZHEIMER, CROSSLINKING)
 10 "BUSH ASHLEY I"/IN
 42377 ALZHEIMER
 3013 ALZHEIMERS

42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
197031 CROSSLINKING
128 CROSSLINKINGS
197086 CROSSLINKING
(CROSSLINKING OR CROSSLINKINGS)
0 ALZHEIMER, CROSSLINKING
(ALZHEIMER (W) CROSSLINKING)
L7 0 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER, CROSSLINKING)

=> S (E3) AND (ALZHEIMER, METALS)
10 "BUSH ASHLEY I"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
855839 METALS
0 ALZHEIMER, METALS
(ALZHEIMER (W) METALS)
L8 0 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER, METALS)

=> S (E3) AND (ALZHEIMER, REDOX)
10 "BUSH ASHLEY I"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
133466 REDOX
8 REDOXES
133469 REDOX
(REDOX OR REDOXES)
0 ALZHEIMER, REDOX
(ALZHEIMER (W) REDOX)
L9 0 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER, REDOX)

=> S (E3) AND (ALZHEIMER)
10 "BUSH ASHLEY I"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
L10 7 ("BUSH ASHLEY I"/IN) AND (ALZHEIMER)

=> E HUANG XUDONG/IN 25
E1 1 HUANG XUANYI/IN
E2 1 HUANG XUBIN/IN
E3 13 --> HUANG XUDONG/IN
E4 1 HUANG XUE F/IN
E5 1 HUANG XUE LIN/IN
E6 2 HUANG XUEBIN/IN
E7 3 HUANG XUECHAO/IN
E8 2 HUANG XUEFEI/IN
E9 1 HUANG XUEFEN/IN
E10 1 HUANG XUEFENG/IN
E11 3 HUANG XUEHONG/IN
E12 1 HUANG XUEHUA/IN
E13 5 HUANG XUEHUI/IN
E14 30 HUANG XUEJIE/IN
E15 1 HUANG XUEJIN/IN
E16 1 HUANG XUEJING/IN
E17 1 HUANG XUEJU/IN
E18 1 HUANG XUEJUE/IN
E19 1 HUANG XUEJUN/IN
E20 1 HUANG XUELIANG/IN
E21 2 HUANG XUELUN/IN
E22 3 HUANG XUEMEI/IN
E23 1 HUANG XUENAN/IN
E24 1 HUANG XUENGUANG/IN
E25 3 HUANG XUEPING/IN

=> S (E3) AND (ALZHEIMER)
13 "HUANG XUDONG"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
L11 7 ("HUANG XUDONG"/IN) AND (ALZHEIMER)

=> DIS L11 1 TI

L11 ANSWER 1 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Amyloid-binding, metal-chelating agents

=> DIS L11 2 TI

L11 ANSWER 2 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method of screening for drugs useful in treating Alzheimer's
disease based on alteration of production of reduced metal ions and hydrogen
peroxide

=> DIS L11 3 TI

L11 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Amyloid-binding, metal-chelating imaging and therapeutic agents

=> DIS L11 4 TI

L11 ANSWER 4 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Methods for identifying an agent that inhibits oxygen-dependent hydrogen
peroxide formation activity but does not inhibit superoxide-dependent
hydrogen peroxide formation

=> DIS L11 5 TI

L11 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Method of screening for drugs useful in treating Alzheimer's
disease

=> DIS L11 6 TI

L11 ANSWER 6 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Agents for use in the treatment of Alzheimer's disease

=> DIS L11 7 TI

L11 ANSWER 7 OF 7 CAPLUS COPYRIGHT 2006 ACS on STN
TI Identification of agents for use in the treatment of Alzheimer's
disease, and methods and compositions for treatment of conditions caused
by amyloidosis and/or A β -mediated ROS formation

=> E ATWOOD CRAIG S/IN 25
E1 2 ATWOOD BRYAN/IN
E2 1 ATWOOD CHARLES T/IN
E3 5 --> ATWOOD CRAIG S/IN
E4 2 ATWOOD DAN/IN
E5 2 ATWOOD DAVID A/IN
E6 2 ATWOOD DAVID ALLAN/IN
E7 3 ATWOOD DONALD K/IN
E8 1 ATWOOD E H/IN
E9 2 ATWOOD EDWARDS S/IN
E10 3 ATWOOD EDWIN H/IN
E11 2 ATWOOD ELBRIDGE L/IN
E12 3 ATWOOD EUGENE R/IN
E13 3 ATWOOD F C/IN
E14 31 ATWOOD FRANCIS C/IN
E15 3 ATWOOD FRANCIS CLARKE/IN
E16 7 ATWOOD GEO E/IN
E17 7 ATWOOD GEORGE E/IN
E18 3 ATWOOD GEORGE F/IN
E19 3 ATWOOD GILBERT R/IN
E20 7 ATWOOD GILBERT RICHARD/IN
E21 2 ATWOOD GLENN A/IN
E22 3 ATWOOD GREG/IN
E23 1 ATWOOD GREGORY/IN
E24 3 ATWOOD GREGORY E/IN
E25 6 ATWOOD HARRY G/IN

=> S (E3) AND (ALZHEIMER)
5 "ATWOOD CRAIG S"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER
(ALZHEIMER OR ALZHEIMERS)
L12 5 ("ATWOOD CRAIG S"/IN) AND (ALZHEIMER)

=> E TANZI RUDOLPH E/IN 25
E1 1 TANZI MARIO/IN
E2 3 TANZI RUDOLPH/IN
E3 28 --> TANZI RUDOLPH E/IN
E4 1 TANZI STEVEN/IN
E5 1 TANZIWA TUNEYUKI/IN
E6 2 TANZILLI JAMES D/IN
E7 3 TANZILLI RICHARD A/IN
E8 1 TANZILLI RICHARD ANTHONY/IN
E9 1 TANZINI SAURO/IN
E10 2 TANZLER RICHARD/IN
E11 8 TANZMAN DANIEL P/IN
E12 1 TANZMAN HERBERT D/IN
E13 1 TANZMANN DANIEL/IN
E14 1 TANZMANN LUBOMIR CS/IN
E15 1 TANZMANN WOLFGANG/IN
E16 1 TANZMEIER PETER/IN
E17 4 TANZO ATSUHARU/IN
E18 10 TANZO JUNJI/IN
E19 1 TANZO TOMOHARU/IN
E20 4 TANZO TOMOJI/IN
E21 1 TANZOLA JOHN C/IN
E22 3 TANZOLA WM A/IN
E23 5 TANZOSH JAMES M/IN
E24 7 TANZYBAEVA L V/IN
E25 1 TANZYBAEVA LYUDMILA V/IN

=> S (E3) AND (ALZHEIMER)

28 "TANZI RUDOLPH E"/IN
42377 ALZHEIMER
3013 ALZHEIMERS
42445 ALZHEIMER

(ALZHEIMER OR ALZHEIMERS)

L13 25 ("TANZI RUDOLPH E"/IN) AND (ALZHEIMER)